Annals of Clinical and Analytical Medicine
Original Research

Relationship between prediabetic conditions and microalbuminuria

Pre-diabetes & Microalbuminuria

Gulbin Seyman Cetınkaya, Yusuf Uzum
Department of Internal Medicine, Katip Celebi University, Ataturk Training and Research Hospital, Izmir, Turkey

Abstract

Aim: Microalbuminuria is an expected predictor of renal and cardiovascular diseases and even mortality. In this study, it was aimed to elucidate the effect of microalbuminuria with albumin creatinine ratio in pre-diabetic patients and to follow patients for a period of 2 years.

Material and Methods: In this prospective study, we have enrolled 90 patients, including n=30 impaired glucose tolerance (IGT), n=30 impaired fasting glucose (IFG), and n=30 in the control group. The diagnosis of IGT has been established via 75 gr glucose loading. The enrolled patients have been followed up every 6 months for a period of 2 years prospectively. The renal function tests, insulin levels, body mass indexes, uric acid levels, CRP levels, and urinary ACR levels were recorded and monitored every 6 months, and annual data were noted. Individuals who developed diabetes during follow-up were recorded and their treatments were initiated.

Results: After a two-year follow-up of the patients, 46.7% (n=14) of patients with impaired glucose tolerance (IGT) had impaired glucose tolerance again, while 13.3% (4 individuals) had impaired fasting glucose and 23.3% (n=7) had normal glucose tolerance and 16.7% (n=5) had diabetes. Similarly, impaired fasting glucose was observed in 66.7% (n=20) of patients with impaired fasting glucose (IFG) at the beginning, while impaired glucose tolerance was observed in 6.7% (n=2) and 23.3% (n=7) had normal glucose tolerance and 3.3% (n=1) had diabetes.

Discussion: Regarding the outcomes of this study, it can be concluded that the prevalence of microalbuminuria has been observed to be higher in pre-diabetic situations such as IFG and IGT. Although both IFG and IGT have the progression potential towards diabetes, IGT should be considered a more serious condition.

Keywords

Microalbuminuria, Pre-Diabetes, Renal Impairment, Diabetic Nephropathy

DOI: 10.4328/ACAM.21506 Received: 2022-11-27 Accepted: 2023-01-13 Published Online: 2023-01-18 Printed: 2023-06-01 Ann Clin Anal Med 2023;14(6):488-493
Corresponding Author: Yusuf Uzum, Department of Internal Medicine, Katip Celebi University, Ataturk Training, and Research Hospital, Izmir, Turkey.
E-mail: yusufuzum@gmail.com P: +90 536 665 87 86 / +90 232 244 44 44
Corresponding Author ORCID ID: https://orcid.org/0000-0001-5027-1827
This study was approved by the Ethics Committee of Izmir Katip Celebi University (Date: 2021-03-24, No: 0121)

Introduction

Diabetes mellitus is an emerging health issue with increasing prevalence all over the world, especially due to the increase in type 2 diabetes (type 2 DM). Diabetic nephropathy is one of the major complications of diabetes that outrageously increases cost of care. As the progression of diabetes continues, diabetic renal disease will account for >50% of patients in dialysis units [1].

Additionally, the prevalence of undiagnosed diabetes or prediabetes is considerably high in chronic renal diseases. Cross-sectional studies show that prediabetes is associated with chronic renal failure (CRF), but it is not known whether it will predict CRF in people who do not progress to diabetes. Prospective studies have not demonstrated that prediabetes is an independent risk factor for albuminuria or the frequency of CRF. It is thought that prediabetes may be a causal factor for the development of CRF [2, 3].

The American Diabetes Association (ADA) recommends 24-hour urine microalbumin testing as the gold standard for nephropathy screening in diabetic patients [4]. However, difficulties in collecting urine for 24 hours showed that spot urine albumin to creatinine ratio (ACR) measured in diabetic patients can be utilized in the detection of nephropathy in diabetic patients [5]. However, the validity of ACR in spot urine in prediabetic patients has not yet been clearly demonstrated. The prevalence of microalbuminuria and macroalbuminuria for both types of diabetes is 30-35% [6].

Albuminuria (albumin to creatinine ratio) or urinary albumin excretion (UAE) should be analyzed 5 years after the diagnosis of type 1 diabetes or at puberty and should be checked at the time of diagnosis and annually thereafter in type 2 diabetes mellitus. The first-morning (or spot) urine is preferred in the analysis of albumin creatinine ratio. It has been stated that rather than progressing to microalbuminuria or macroalbuminuria, the change in UAE must be taken into account [7].

Normoalbuminuria is defined as <30mg/g ACR in the first-morning (or spot) urine and <30 mg/day UAE, <20 µg/minutes UAE rate in a 24-hour period. Microalbuminuria is defined as 30 – 300 mg/g ACR in the first-morning (or spot) urine and 30 – 300 mg/day UAE, 20 – 200 µg/minutes UAE rate in a 24-hour period. Macroalbuminuria or clinical albuminuria is defined as >300 mg/g ACR in the first-morning (or spot) urine and >300 mg/day UAE, >200 µg/minutes UAE rate in a 24-hour period. The clinician should bear in mind that heavy exercise, infection, fever, congestive heart failure, hypertension, and remarkable hyperglycemia might elevate UAE. Nephropathy screening tests should not be performed until these problems are solved or controlled. In order to establish a diagnosis of MAU 2 of 3 laboratory results should indicate higher values [8].

Melsom et al. (2016) reported 1261 nondiabetic patients followed for 5.6 years [9]. In their study, prediabetes was also observed in the early stages of diabetes as well as glomerular hyperfiltration and albuminuria have been shown to be associated with the development of diabetes [9]. The measurement of microalbuminuria to investigate early-stage nephropathy in adults eGFR needs to be calculated. In addition, eGFR can be calculated from the MDRD, CKD-EPI, or Cockcroft-Gault formulas by measuring the serum creatinine level [9, 10].

To monitor the progression of diabetic nephropathy in patients who develop microalbuminuria, the urine albumin/creatinine ratio should be measured more frequently. Nephropathy is one of the most important causes of morbidity and mortality in adult diabetic patients. Patients with persistent microalbuminuria have a high risk of retinopathy, therefore fundus examination should be performed more closely [11].

The inhibition of the renin angiotensin system plays a major role in the prevention of glomerular hypertension and hyperfiltration thus reducing albuminuria and kidney damage. These renal protective effects are mainly due to the vasodilator effect on efferent glomerular arterioles and as a result attributed to a decrease in glomerular pressure. In patients with microalbuminuria, it is appropriate to use an angiotensin-converting enzyme (ACE) inhibitor or angiotensin receptor blocker (ARB) to help prevent progression to macroalbuminuria [12].

Glycemic and blood pressure control, treatment of dyslipidemia, and smoking cessation are essential to prevent the progression of nephropathy. In addition, weight loss may play an important role in preventing DRD and slowing its progression. Renal replacement therapy should be applied when end-stage renal failure develops [13].

In this study, it was aimed to elucidate the effect of microalbuminuria with albumin creatinine ratio in pre-diabetic patients and to follow the patients for a period of 2 years. In this follow-up period, patients with microalbuminuria will be compared with pre-diabetic patients without microalbuminuria and it will be investigated whether microalbuminuria is a risk factor in pre-diabetic patients.

Material and Methods

In this prospective study, we have enrolled 90 patients, including n=30 patients with impaired glucose tolerance (IGT), n=30 patients with impaired fasting glucose (IFG), and n=30 in the control group. The diagnosis of IGT has been established via 75 gr glucose loading in individuals who have been admitted to the outpatient clinic of our institution. The study has been approved by the Ethics Committee and was conducted according to the Guidelines of the Declaration of Helsinki. Informed consent has been obtained from all participants.

The enrolled patients have been followed up every 6 months for a period of 2 years prospectively. The renal function tests, insulin levels, body mass indexes, uric acid levels, CRP levels, and urinary ACR levels were recorded and monitored every 6 months, and annual data were noted. Individuals who developed diabetes during follow-up were recorded and their treatments were initiated.

Inclusion Criteria

The inclusion criteria were as follows: results of glucose tolerance test (GTT, 75 g), and IFG≥ 100mg/dL. IGT was defined as between 140-199 mg/dL of serum glucose after two-hour oral 75 g glucose consumption.

Exclusion Criteria

The exclusion criteria were hypertension, heart failure, patients with overt diabetes that would cause microalbuminuria, renal failure, pre-existing proteinuria, recent urinary tract infection. Patients who had been using corticosteroids, spironolactone,

ACE inhibitors, and ARBs were excluded from the study.

Statistical Analysis

Patient data collected within the scope of the study were analyzed with the IBM Statistical Package for the Social Sciences (SPSS) for Windows 23.0 (IBM Corp., Armonk, NY) package program. Frequency and percentage were given for categorical data, and median, minimum, and maximum descriptive values for continuous data. For comparisons between groups, the Mann Whitney U-Test was used for two groups, the Kruskal Wallis H-Test for more than two groups, and the Pearson Chi-square Test was used to compare categorical variables. The results were considered statistically significant when the p-value was less than 0.05.

Ethical Approval

Ethics Committee approval for the study was obtained.

Results

Within the scope of the study, the evaluation was made on 90 patients, including n=30 impaired glucose tolerance (IGT), n=30 impaired fasting glucose (IFG), and n=30 in the control group. According to the initial evaluations, there was no statistically significant difference between the three groups in terms of gender (p>0.05). Age, body mass index (BMI), initial laboratory parameters of FBG, PPBG, HBA1C, insulin, BUN, uric acid, urine albumin, urinary creatinine, ACR, and CRP values were found statistically significant between the groups (p<0.05) (Table 1). When the age distribution of the groups, BMI, FBG, HBA1C, insulin, BUN, uric acid, and CRP values were examined, it was seen that the values were higher in the IGT and IFG groups compared to the control group.

The demographic, baseline, and second-year laboratory parameters distribution of 30 patients with impaired glucose tolerance (IGT) and 30 patients with impaired fasting glucose (IFG) included in the study are shown in Table 2. When the table was examined, it was seen that there was a statistically significant difference between the two groups in terms of demographic data, age, and BMI (p<0.05). There was a significant difference between the two groups in PPBG, HBA1C, insulin, urine albumin and ACR parameters (p<0.05).

It was noteworthy that the initial laboratory results of patients with impaired glucose tolerance were higher than those of individuals with impaired fasting glucose. In the second-year laboratory data, significant differences were observed in other parameters other than urine albumin, which were significant in the initial laboratory data such as PPBG, HBA1C, insulin, urine albumin, and ACR (p<0.05). It was observed that the laboratory results of patients with impaired glucose tolerance were higher than the laboratory results of individuals with impaired fasting glucose. These parameters showed significant differences in laboratory parameters in the second year.

After a two-year follow-up of the patients, 46.7% (n=14) of patients with impaired glucose tolerance (IGT) had impaired glucose tolerance again, while 13.3% (4 individuals) had impaired fasting glucose and 23.3% (n=7) had normal glucose tolerance and 16.7% (n=5) had diabetes. Similarly, impaired fasting glucose was observed in 66.7% (n=20) of patients with

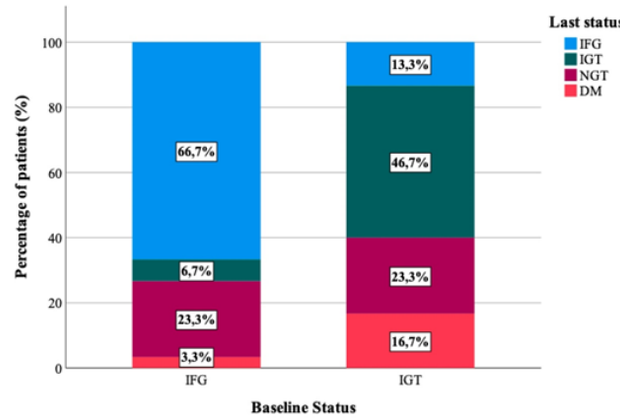

Figure 1. Distribution of final status of treatment groups relative to baseline.

Table 1. Evaluation of demographic and baseline parameters of treatment groups and control groups.

Characteristics (N=90)	Total	IFG (n=30)	IGT (n=30)	Control (n=30)	P-value
	N (%) or Median (Min-Max)	N (%) or Median (Min-Max)	N (%) or Median (Min-Max)	N (%) or Median (Min-Max)	
Gender					
Female	52 (57.8)	17 (56.7)	19 (63.3)	16 (53.3)	0,727
Male	38 (42.2)	13 (43.3)	11 (36.7)	14 (46.7)	
Age, years	45 (20-58)	50 (32-58)	45 (32-57)	36 (20-58)	<0.001
BMI	24.9 (21.2-42)	25.1 (22.9-31.3)	27.4 (22.6-42)	23.9 (21.2-24.9)	<0.001
Initial FBG	104 (75-123)	106 (100-122)	105.5 (101-123)	92.5 (75-99)	<0.001
Initial PPBG	122 (77-175)	104 (77-147)	149 (89-175)	119 (95-136)	<0.001
Initial HBA1C	5.6 (4.2-6.4)	5.6 (4.5-6.4)	5.7 (5.2-6.4)	5.2 (4.2-5.8)	<0.001
Initial Insulin	9.5 (1.6-40.6)	9.9 (3.6-23.2)	13.6 (3.8-40.6)	6.2 (1.6-15.2)	<0.001
Initial BUN	12 (6-32)	13 (9-21)	12 (9-32)	11.5 (6-18)	0.050
Initial Creatinine	0.8 (0.5-72)	0.8 (0.6-1.1)	0.7 (0.5-72)	0.8 (0.5-1)	0.216
Initial Uric Acid	4.7 (3-8.7)	4.9 (3-8.7)	4.7 (3.4-6.7)	4.2 (3-7.6)	0.033
Initial Urine Albumin	12.5 (6-157)	8 (6.8-16)	12 (6.8-157)	17 (6-31)	<0.001
Initial Urine Creatinine	127 (36-341)	130 (36-327)	111 (40-341)	148.5 (36-244)	0.011
Initial ACR	105.3 (39-1635)	69.1 (39-188.8)	125.5 (44-1635)	121 (58-389)	<0.001
Initial CRP	0.12 (0.03-4.2)	0.2 (0.03-4.2)	0.22 (0.03-2.88)	0.10 (0.04-0.21)	0.002

Table 2. Evaluation of demographic and follow-up parameters of treatment groups.

Characteristics (N=60)	Total	IFG (n=30)	IGT (n=30)	P-value
	N (%) or Median (Min-Max)	N (%) or Median (Min-Max)	N (%) or Median (Min-Max)	
Gender				
Female	36 (60.0)	17 (56.7)	19 (63.3)	0.792
Male	24 (40.0)	13 (43.3)	11 (36.7)	
Age, years	47 (32-58)	50 (32-58)	45 (32-57)	0.010
BMI	26.2 (22.6-42)	25.1 (22.9-31.3)	27.4 (22.6-42)	<0.001
Initial FBG	106 (100-123)	106 (100-122)	105.5 (101-123)	0.784
Initial PPBG	125.5 (77-175)	104 (77-147)	149 (89-175)	<0.001
Initial HBA1C	5.6 (4.5-6.4)	5.6 (4.5-6.4)	5.7 (5.2-6.4)	0.005
Initial Insulin	11.5 (3.6-40.6)	9.9 (3.6-23.2)	13.6 (3.8-40.6)	<0.001
Initial BUN	13 (9-32)	13 (9-21)	12 (9-32)	0.355
Initial Creatinine	0.7 (0.5-72)	0.8 (0.6-1.1)	0.7 (0.5-72)	0.081
Initial Uric Acid	4.8 (3-8.7)	4.9 (3-8.7)	4.7 (3.4-6.7)	0.717
Initial Urine Albumin	10 (6.8-157)	8 (6.8-16)	12 (6.8-157)	0.014
Initial Urine Creatinine	114.5 (36-341)	130 (36-327)	111 (40-341)	0.115
Initial ACR	86 (39-1635)	69.1 (39-188.8)	125.5 (44-1635)	0.001
Initial CRP	0.2 (0.03-4.2)	0.2 (0.03-4.2)	0.22 (0.03-2.88)	0.865
FBG (2 years)	106.5 (92-142)	106 (92-128)	106.5 (93-142)	0.739
PPBG (2 years)	133 (69-215)	121.5 (69-163)	149 (89-215)	<0.001
HBA1C (2 years)	5.7 (4.7-7.3)	5.7 (5.1-6.6)	5.9 (4.7-7.3)	0.019
Insulin (2 years)	11.1 (3.6-37)	11 (3.6-17)	12.8 (5.4-37)	0.044
BUN (2 years)	12 (10-22)	12 (11-22)	12 (10-22)	0.232
Creatinine (2 years)	0.7 (0.5-1.1)	0.7 (0.5-1)	0.7 (0.6-1.1)	0.603
Uric Acid (2 years)	5.2 (3.4-7.6)	5.2 (3.4-7.2)	5.4 (4-7.6)	0.717
Urine Albumin (2 years)	11 (6-153)	9 (6-25)	13 (6.8-153)	0.051
Urine Creatinine (2 years)	116 (48-228)	119 (52-228)	111 (48-186)	0.263
ACR (2 years)	101.5 (40.5-3187)	81.5 (40.5-215)	115 (55-3187)	0.004
CRP (2 years)	0.2 (0-2.3)	0.2 (0-1.1)	0.2 (0-2.3)	0.756
DM Final Status				
IFG	24 (40.0)	20 (66.7)	4 (13.3)	<0.001
IGT	16 (26.7)	2 (6.7)	14 (46.7)	
NGT	14 (23.3)	7 (23.3)	7 (23.3)	
DM	6 (10.0)	1 (3.3)	5 (16.7)	

impaired fasting glucose (IFG) at the beginning, while impaired glucose tolerance was observed in 6.7% (n=2) and 23.3% (n=7) had normal glucose tolerance and 3.3% (n=1) had diabetes (Figure 1).

Discussion

The prevalence of impaired glucose tolerance worldwide has been stated as 6.7% in the International Diabetes Federation (IDF) atlas (2015). It is estimated that 318 million people are presumed to be pre-diabetic and this number is predicted to reach 481 million people in 2040. The frequency of pre-diabetes increases with age. The main differences between IFG and IGT can be elaborated as IGT is associated with peripheral insulin resistance (skeletal muscle), while IFG is associated with increased gluconeogenesis. In combined situations, hepatic and extrahepatic insulin resistance and increased gluconeogenesis persist. Isolated first phase insulin secretion defect (early phase) is observed in IFG, while both the first (late phase) and the second phase release defect is associated with IGT [14]. IGT is more closely related to future diabetes and the coexistence of IFG and IGT doubles the risk. IGT has been correlated with increased cardiovascular risks and microvascular complications such as retinopathy and neuropathy [15].

The rate of progression to diabetes from IFG and IGT within 3-5 years is 25% [16]. On the other hand, glucose tolerance remains the same in 50% of the individuals, while 25% returns to normal. Those with additional diabetes clinical risk factors (obesity, family history) have a higher risk of developing diabetes. Additionally, the annual risk of conversion of impaired glucose tolerance to diabetes is 3-11%, while the lifetime risk of type 2 diabetes varies up to 50% [17]. The conversion of pre-diabetes to diabetes in different genetic characteristics is reported as 114.4 versus 2.3/1000 people [18]. In our study half of the patients (46.7%) with IGT had no change in their status in the second year, while 16.7 of them have progressed to diabetes. Additionally, 66.7% of patients with impaired fasting glucose (IFG) at the beginning remained the same within 2 years, while impaired glucose tolerance was observed in 6.7%, 23.3% had normal glucose tolerance and only 3.3% had diabetes.

Prediabetes is a major risk factor for diabetic chronic renal failure (CRF). The vast majority of newly diagnosed diabetic

patients will develop CRF despite optimal treatment. Pre-diabetes is about twice as common as diabetes, affects 20-35% of adults, and progresses to diabetes after 10 years in approximately 45-50% of individuals. Cross-sectional studies show that prediabetes is associated with CRF, but it is not known whether it will predict CRF in people who do not progress to diabetes.

Previous literature has reported that approximately 1/3 of newly diagnosed diabetics already have kidney damage, indicating a very early onset of renal failure. In the meta-analysis by Echouffo-Tcheugui et al. (2016), 9 cohort studies evaluating a total of 185.452 patients, the relative risk for the development of CRF in prediabetics was found to be 1.11 (CI 951.02 -1.21) [19].

In this current study, we have investigated the effect of microalbumin¬uria with albumin to creatinine ratio in pre-diabetic patients and followed up the patients for a period of 2 years with respect to control group. In this follow-up period, patients with microalbuminuria were compared with pre-diabetic patients without microalbuminuria, and whether microalbuminuria is a risk factor in pre-diabetic patients was investigated.

Similar studies have been published previously elaborating microalbu¬minuria in prediabetic subjects. Franciosi et al. found that microalbuminuria was present in 6.9%, 5.6%, and 4.3% in IFG, IGT, and control groups, re¬spectively [20]. Tapp et al. have reported that the prevalence of microalbuminuria was 8.3% in IFG, 9.9% in IGT, and 4.3% in the control group [21]. It should be kept in mind that different values could be attributed to geographical demographics and laboratory methods. However, it is evident that urine albumin was significantly higher in patients with IFG and IGT.

The association between IFG and microal¬buminuria has been introduced by Meigs in Framingham Offspring Study (2002) two decades ago [22]. Bahar et al. (2013) published an important article emphasizing that the prevalence of microal¬buminuria in the IGT group was higher than in the IFG group, and IGT was identified as the most important risk factor for microalbuminuria [23]. Our results were in line with these studies and we have found that the laboratory parameters of patients with impaired glucose tolerance were higher than those of individuals with impaired fasting glucose. These differences such as PPBG, HBA1C, insulin, urine albumin, and ACR were significantly persistent in the second year. It was observed that the laboratory results of patients with impaired glucose tolerance were higher than the laboratory results of individuals with impaired fasting glucose. These parameters showed significant differences in laboratory parameters in the second year.

The main limitation of this study could be attributed to its relatively small sample size. Additionally, the ideal follow-up period should have been at least 5 years in order to accurately observe the development of diabetes and microalbuminuria in renal functions. All the individuals have been performed OGT at the diagnosis stage however, this test had not been re-performed annually.

Since there are few studies emphasizing renal dysfunction that can develop in prediabetic patients, this may be an advantage of this research. Increasing the widespread use of microalbuminuria for the predictive purpose was the main focus of this study leveraging a cost-effective test that can be performed in each hospital in the detection of renal injury that may develop in prediabetic patients. In addition, as in other studies requiring follow-up, the fact that during the two years of follow-up period enabled patients to display a more conscious and careful attitude may be another strength of this study.

Conclusion

Regarding the outcomes of this study, one can conclude that the prevalence of microalbuminuria has been observed higher in pre-diabetic situations such as IFG and IGT. Although both IFG and IGT have the progression potential towards diabetes, IGT is considered a more serious condition.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

Funding: *None*

Conflict of interest

None of the authors received any type of financial support that could be considered potential conflict of interest regarding the manuscript or its submission.

References

1. Narayan S, Lakshmipriya N, Vaidya R, Bai MR, Sudha V, Krishnaswamy K, et al. Association of dietary fiber intake with serum total cholesterol and low density lipoprotein cholesterol levels in Urban Asian-Indian adults with type 2 diabetes. Indian J Endocrinol Metab. 2014;18(5):624-30.

2. Yao B, Fang H, Xu W, Yan Y, Xu H, Liu Y, et al. Dietary fiber intake and risk of type 2 diabetes: a dose-response analysis of prospective studies. Eur J Epidemiol. 2014; 29(2): 79–88.

3. Emadian A, Thompson J. A Mixed-Methods Examination of Physical Activity and Sedentary Time in Overweight and Obese South Asian Men Living in the United Kingdom. Int J Environ Res Public Health. 2017;14(4): 348.

4. American Diabetes Association. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2019. Diabetes care. 2019; 42(Suppl. 1): S13–28.

5. Lee IM, Shiroma EJ, Lobelo F, Puska P, Blair SN, Katzmarzyk PT, et al. Effect of physical inactivity on major non-communicable diseases worldwide: An analysis of burden of disease and life expectancy. Lancet. 2012; 380(9838):219-29.

6. Einarson TR, Bereza BG, Acs A, Jensen R. Systematic literature review of the health economic implications of early detection by screening populations at risk for type 2 diabetes. Curr Med Res Opin. 2017;33(2):331-58.

7. Iliodromiti S, Ghouri N, Celis-Morales CA, Sattar N, Lumsden MA, Gill JM. Should physical activity recommendations for South Asian adults be ethnicity-specific? Evidence from a cross-sectional study of South Asian and white European men and women. PLoS One.2016;11(8):e0160024.

8. Hameed UA, Manzar D, Raza S, Shareef MY, Hussain ME. Resistance training leads to clinically meaningful improvements in control of glycemia and muscular strength in untrained middle-aged patients with type 2 diabetes mellitus. N Am J Med Sci. 2012;4(8):336-43.

9. Melsom T, Schei J, Stefansson VTN, Solbu MD, Jenssen TG, Mathisen UD, et al. Prediabetes and Risk of Glomerular Hyperfiltration and Albuminuria in the General Nondiabetic Population: A Prospective Cohort Study. Am J Kidney Dis. 2016;67(6):841-50.

10. Green R, Milner J, Joy EJ, Agrawal S, Dangour AD. Dietary patterns in India: A systematic review. Br J Nutr. 2016;116(1):142-8.

11. Becerra-Tomás N, Díaz-López A, Rosique-Esteban N, Ros E, Buil-Cosiales P, Corella D, et al. Legume consumption is inversely associated with type 2 diabetes incidence in adults: A prospective assessment from the PREDIMED study. Clin Nutr. 2018;37(3):906-13.

12. Narasimhan S, Nagarajan L, Vaidya R, Gunasekaran G, Rajagopal G, Parthasarathy V, et al. Dietary fat intake and its association with risk of selected components of the metabolic syndrome among rural South Indians. Indian J Endocrinol Metab. 2016;20(1):47-54.

13. Lakshmipriya N, Gayathri R, Praseena K, Vijayalakshmi P, Geetha G, Sudha V, et al. Type of vegetable oils used in cooking and risk of metabolic syndrome among Asian Indians. Int J Food Sci Nutr. 2013;64(2):131-9.
14. Nigam P, Bhatt S, Misra A, Chadha DS, Vaidya M, Dasgupta J, et al. Effect of a 6-month intervention with cooking oils containing a high concentration of monounsaturated fatty acids (olive and canola oils) compared with control oil in male Asian Indians with nonalcoholic fatty liver disease. Diabetes Technol Ther. 2014;16(4):255-61.
15. Gulati S, Misra A. Abdominal obesity and type 2 diabetes in Asian Indians: Dietary strategies including edible oils, cooking practices and sugar intake. Eur J Clin Nutr. 2017;71(7):850-7.
16. Gulati S, Misra A, Sharma M. Dietary fats and oils in India. Curr Diabetes Rev. 2017;13(5):438-43.
17. Bhardwaj S, Passi SJ, Misra A, Pant KK, Anwar K, Pandey RM, et al. Effect of heating/reheating of fats/oils, as used by Asian Indians, on trans fatty acid formation. Food Chem. 2016; 212:663-70.
18. Kakde S, Bhopal RS, Bhardwaj S, Misra A. Urbanized South Asians' susceptibility to coronary heart disease: The high-heat food preparation hypothesis. Nutrition. 2017; 33:216-24.
19. Echouffo-Tcheugui JB, Narayan KM, Weisman D, Golden SH, Jaar BG. Systematic review or meta-analysis association between prediabetes and risk of chronic kidney disease: a systematic review and meta-analysis. Diabet Med. 2016;33(12):1615-24.
20. Franciosi M, Pellegrini F, Sacco M, De Berardis G, Rossi MCE, Strippoli GFM, et al. on behalf of the IGLOO (Impaired Glucose tolerance, and Long-term Outcomes Observational Study) Study Group: Identifying patients at risk for microalbuminuria via in¬teraction of the components of the metabolic syndrome: A cross-sectional analytic study. Clin J Am Soc Nephrol. 2007;2(5):984-91.
21. Tapp RJ, Shaw JE, Zimmet PZ, Balkau B, Chadban SJ, Tonkin AM, et al. Albuminuria is evident in the early stages of diabetes onset: results from the Australian Diabetes, Obesity, and Lifestyle Study (AusDiab). Am J Kidney Dis.2004;44(5):792-8.
22. Meigs JB, D'Agostino RB, Sr, Nathan DM, Rifai N, Wilson PW. Lon¬gitudinal association of glycemia and microalbuminuria: the Framingham Offspring Study. Diabetes Care. 2002;25(6):977-83.
23. Bahar A, Makhlough A, Yousefi A, Kashi Z, Abediankenari S. Correlation between prediabetes conditions and microalbuminuria. Nephrourol Mon. 2013;5(2):741-4.

How to cite this article:
Gulbin Seyman Cetınkaya, Yusuf Uzum. *Relationship between prediabetic conditions and microalbuminuria. Ann Clin Anal Med 2023;14(6):488-493*

This study was approved by the Ethics Committee of Izmir Katip Celebi University (Date: 2021-03-24, No: 0121)